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Approach bias modification training in bulimia nervosa and binge eating disorder: a pilot randomised controlled trial

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RUNNING HEAD: APPROACH BIAS MODIFICATION IN BULIMIC EATING DISORDERS

Abstract

Objective: Bulimia nervosa (BN) and binge eating disorder (BED) are associated with poorly controlled approach behaviour towards food resulting in binge eating. Approach bias modification (ABM) may reduce these automatic action tendencies (i.e., approach bias) towards food and may thus decrease binge eating and related symptoms. **Method:** A total of 56 patients with BN/BED participated in this double-blind, randomised controlled trial (RCT) comparing real and sham ABM. The real ABM condition adopted an implicit learning paradigm in which participants were trained to show avoidance behaviour in response to food cues. Participants in the sham condition used a similar task but were not trained to avoid food cues. Both conditions comprised 10 training sessions within 4 weeks. **Results:** Participants in both groups experienced significant reductions in binge eating, eating disorder symptoms, trait food craving, and food cue reactivity. Real ABM tended to result in greater reductions in eating disorder symptoms than sham ABM. Food intake, approach bias, and attention bias toward food did not change. **Discussion:** This is the first RCT on ABM in eating disorders. The findings provide limited support for the efficacy of ABM in BN/BED and pose questions regarding its active ingredients and its usefulness as a stand-alone treatment for eating disorders.

Keywords: Cognitive bias modification, eating disorders, treatment, information processing

Approach bias modification training in bulimia nervosa and binge eating disorder: a randomised controlled trial

Introduction

Binge eating is the hallmark of Bulimia Nervosa (BN) and Binge Eating Disorder (BED) (APA, 2013). Dual-process models suggest that two information processing systems contribute to it: an impulsive system operating rapidly and automatically, and a reflective system operating slower and more deliberately (Bechara, 2005; Wiers, Gladwin, Hofmann, Salemink, & Ridderinkhof, 2013). In the impulsive system, food cues are evaluated primarily regarding their current emotional and motivational significance. In contrast, the reflective system involves higher-order processes of cognitive control and value-based decision-making that take long-term consequences into account. In addictive disorders, the impulsive system is considered to dominate to a large degree the initiation of addictive behaviour, while the reflective system lacks power to control this behaviour (Wiers et al., 2013). Correspondingly, BN and BED are associated with strong impulsive responses and poor cognitive control towards food cues (Wu, Hartmann, Skunde, Herzog, & Friederich, 2013). Previous research has shown that eating disorders (ED) and unhealthy eating behaviour are linked to cognitive biases that are considered to primarily operate in the impulsive system, particularly by increasing attention and approach tendencies towards food (Brignell, Griffiths, Bradley, & Mogg, 2009; Brockmeyer, Hahn, Reetz, Schmidt, & Friederich, 2015b; Brooks, Prince, Stahl, Campbell, & Treasure, 2011; Havermans, Giesen, Janneke C. A. H., Houben, & Jansen, 2011; Kemps, Tiggemann, Martin, & Elliott, 2013; Veenstra & Jong, 2010).

Traditional psychotherapy programmes may not be best suited to directly alter such cognitive biases (Renwick, Campbell, & Schmidt, 2013; Wiers et al., 2013). Instead, cognitive bias modification programmes are intended to target these biases more directly. Especially in addictions, approach bias modification (ABM), a variant of cognitive bias modification, has been shown to reduce automatic action tendencies towards alcohol cues and relapse rates (Eberl et al., 2013; Wiers, Eberl, Rinck, Becker, & Lindenmeyer, 2011). Recent studies also showed that this intervention reduced approach bias towards food cues and actual food intake in student populations (Dickson, Kavanagh, & MacLeod, 2016; Schumacher, Kemps, & Tiggemann, 2016).

In a proof-of-concept study (Brockmeyer, Hahn, Reetz, Schmidt, & Friederich, 2015a) we examined ABM in individuals with high levels of trait food craving. In this uncontrolled study, we found significant reductions in the targeted approach bias towards food as well as in attentional bias towards food, trait and cue-elicited food craving, and ED symptoms, with medium to large effect sizes. Whilst effective

treatment for BN and BED exists in the form of disorder-focused cognitive behavioural therapy (NICE, 2017), this approach is not effective for all patients. Thus, novel (add-on) treatment approaches, especially those that are easily disseminated, efficient, and cost-effective, are urgently needed (Schmidt & Campbell, 2013; Treasure, Claudino, & Zucker, 2010).

This is the first clinical trial testing ABM in BN/BED. The central aim of this randomised controlled pilot trial was to examine whether ABM reduces binge eating in BN and BED patients. More precisely, BN/BED patients who received real ABM were expected to show greater reductions in the number of objective binge eating episodes (OBEs) than patients who received sham ABM. Furthermore, we examined whether real ABM would also lead to greater reductions in ED symptoms, trait food craving, food cue reactivity, food intake in the laboratory, and approach and attention bias towards visual food stimuli than sham ABM.

Method

Study design

This study was a multicentre, double-blind, randomised controlled superiority trial adopting a parallel-group design. The trial protocol has been published elsewhere (Brockmeyer, Schmidt, & Friederich, 2016). The trial was registered in the *German Clinical Trials Register* (DRKS00010231). Independent research ethics committees at every participating centre approved the study protocol. Participants with BN or BED were randomly allocated to receive either 10 sessions of real ABM or sham ABM. Participants were recruited from three different sites (Heidelberg and Düsseldorf, Germany; London, UK). Outcomes were assessed at baseline, post-treatment, and 2-months follow-up.

Participants

Participants (N = 56) were recruited from outpatient units of the Department of General Internal Medicine and Psychosomatics at University Hospital Heidelberg, the South London and Maudsley NHS Foundation Trust's Specialist Adult Eating Disorders Unit, and through websites, circular mails, advertising posters, and adverts in local and social media. Participants received financial compensation for their participation in the study. Written informed consent was obtained from all participants.

Women and men were eligible for participation if they were at least 18 years old and met the criteria of a DSM-5 diagnosis of BN or BED (APA, 2013). Exclusion criteria were: (a) age < 18 years; (b) medical (e.g., electrolyte abnormalities) or psychiatric (e.g., acute suicidality) instability; (c) need for immediate inpatient treatment; (d) lifetime diagnosis of substance dependence, psychosis, bipolar disorder, or borderline personality disorder; (e) psychotropic medication other than selective serotonin reuptake inhibitors (SSRI; patients had to be on a stable medication, i.e., at least 14 days of

SRRI during participation); (f) severe learning disability impeding participants' ability to complete study assessments/treatment; (g) inability to speak fluent English/German (depending on study site).

A total sample size of n=34 participants would have 80% power to detect a medium effect size in a 2 x 2 mixed ANOVA with two groups and two measurements, with a 0.05 two-tailed significance level. We assumed the attrition to follow-up to be up to 25% and that up to 10% of data might get lost due to technical errors (i.e., a=35%). We applied an attrition correction factor of 1/(1-a), and thus needed a sample size of at least *n*=53 participants. To be more conservative, we rounded this up to *n*=56 (i.e., *n*=28 per group).

Six participants dropped out from treatment (i.e., 11%). Participants who received at least 7 of the 10 scheduled training sessions were considered treatment completers. Thus, the treatment completer sample comprised 24 participants in the real ABM condition and 26 participants in the sham ABM condition (i.e., *N*=50 in total).

Procedure

Potential participants were referred to the study by their clinician or self-referred. Study assessors screened participants for eligibility using the *Structured Clinical Interview for DSM Disorders* (SCID) (First, Williams, Spitzer, & Gibbon, 2007; Wittchen, Zaudig, & Fydrich, 1997), a short inclusion/exclusion screen specific to this study, and an assessment of medical and psychiatric history, and medication dosage and stability.

People deemed eligible to take part proceeded to the baseline assessment, where binge-eating frequency and ED symptoms were assessed along with trait food craving, food cue reactivity, food intake, and approach and attention bias towards visual food cues. In order to minimise confounding effects of hunger and satiety on eating-related outcomes, participants were asked to not drink or eat anything other than water in the two hours before the assessment.

Participants were then randomly allocated to the treatment group (real ABM) or control group (sham ABM). Randomisation was performed independently from the trial team by a central study office at Heidelberg University Hospital using randomisation software (RANDI2). Randomisation was stratified for BN/BED diagnosis in a 1:1 ratio. Participants and assessors administering the Eating Disorder Examination interview were 'blinded' to treatment allocation throughout.

The first training session took place on the day of the baseline assessment. Training consisted of 10 sessions over 4 weeks. After the final session, the post-treatment assessment took place and included the same elements as the baseline assessment (again, participants were asked to refrain from eating and drinking - except water - two hours prior to the assessment) except for the interview-based assessment of ED symptoms which were re-assessed at follow-up (two months after treatment termination). To assess whether allocation concealment was successful, participants were asked to

guess their treatment allocation at treatment termination and to indicate how certain they were of this guess. Finally, participants were debriefed and un-blinded to group allocation upon completion of the follow-up, and participants in the sham group were offered the opportunity to receive real ABM after the end of follow-up.

Measures

Screening measures

Participants' body weight and height were objectively obtained by the assessors. The screening module of the SCID (First et al., 2007; Wittchen et al., 1997) was used as a diagnostic screen to identify the presence of psychiatric comorbidities.

Primary outcome measure

The primary outcome variable in this study was change from baseline to follow-up in the number of objective binge eating episodes (OBEs) as assessed by the *Eating Disorder Examination* (EDE) (Cooper & Fairburn, 1987; Hilbert & Tuschen-Caffier, 2016). The EDE is a reliable and valid measure of ED symptoms and is considered the instrument of choice for the assessment and diagnosis of EDs according to the DSM (Cooper & Fairburn, 1987). All assessors were trained in the application of the EDE interview.

Secondary outcome measures

ED symptoms were also assessed by the EDE interview (Cooper & Fairburn, 1987; Hilbert & Tuschen-Caffier, 2016). Further to OBEs and number of days with OBEs ('binge-days'), the EDE also assesses dietary restraint, eating concern, weight concern, and shape concern over the last 28 days. Responses are rated on a 6-point Likert scale, higher scores indicate greater ED symptom severity. Reliability and validity of the interview has been demonstrated in previous studies (Cooper & Fairburn, 1987; Hilbert & Tuschen-Caffier, 2016).

Trait food craving was assessed by the *Food Cravings Questionnaire Trait Version* (Cepeda-Benito, Gleaves, Williams, & Erath, 2000; Meule, Lutz, Vögele, & Kübler, 2012). This self-rating questionnaire consists of 39 items and assesses food craving across nine subscales (e.g., preoccupation with food, anticipation of positive reinforcement that may result from eating). Responses are made on a 6-point Likert scale ranging from 1 ("never") to 6 ("always"). Higher scores indicate stronger trait food craving. Previous research has shown the scale's reliability and validity (Cepeda-Benito et al., 2000; Meule et al., 2012).

Food cue reactivity was assessed as follows: Participants first completed the *Food Cravings Questionnaire-State Version* (Cepeda-Benito et al., 2000; Meule et al., 2012). Following this, food

craving was induced by presenting a five-minute video depicting a range of high-calorie, palatable foods. The food stimuli shown in this video have been rated as being highly appetising and to significantly increase hunger (Kekic et al., 2017). Subsequently, participants completed the *Food Cravings Questionnaire-State Version* again. The increase in state food craving from prior to after watching the video was used as an indicator for food cue reactivity. We previously used the same procedure in two studies on approach bias towards food (Brockmeyer et al., 2015b, 2015a).

Food intake was measured by a *Bogus Taste Test* (Robinson et al., 2017): Participants were instructed to rate three bowls of palatable food items (chocolate, crisps, fruit gums) in terms of their visual attractiveness, smell, and taste. They were told that they were free to eat as much of the offered items as they liked. During the test they were left alone in the room. To minimise demand effects, they were told (beforehand) to throw food which they did not eat into a (prepared) rubbish bin (for health and safety reasons). Consumption was determined after participants had left, by collecting and sorting discarded food from the prepared bin and weighing the bowls both before and after the taste test.

Approach bias towards food (the target cognitive mechanism of the intervention) was measured by the assessment version of the *Approach-Avoidance Task* (AAT) (Rinck & Becker, 2007). In this task, participants were shown colour photographs of high-calorie, palatable foods such as chocolate, chips, and pizza, and non-food (household and office articles such as scrubbers, sponges, and staplers) items on a computer-screen (we have used the same set of pictures in previous studies on BN/BED, Brockmeyer et al., 2015b, 2015a; Meule et al., 2018). They were required to pull or push a joystick in response to the outline of the picture (round vs. rectangular), irrespective of picture content. Format movement assignments were counterbalanced among participants (i.e., half pushed round pictures and half pushed rectangular pictures). When the joystick was pulled, the picture grew bigger and when it was pushed it grew smaller. This zooming-in and zooming-out emphasizes respective sensations of approaching and avoiding and thus combines the proprioceptive (arm movement) and exteroceptive (zooming feature) cues of approach and avoidance behaviour (Neumann & Strack, 2000). The assessment version of the AAT consisted of 80 trials (i.e., 40 food pictures and 40 non-food pictures). The task was performed using Inquisit 4 (Millisecond Software). To determine the approach bias towards food, a compatibility score was calculated by subtracting the median reaction times (RTs) of compatible trials (i.e., RT pull food+RT push non-food) from median RTs of incompatible trials (i.e., RT push food+RT pull non-food) (Becker et al., 2016; Vrijsen et al., 2018). Thus, a positive value denotes a food-specific approach bias, whereas a negative value indicates a food-specific avoidance bias.

Attention bias towards food was measured using a dot-probe task (Miller & Fillmore, 2010). After presenting a fixation point in the centre of the screen (500ms), a pair of pictures (food and non-food) appeared simultaneously for 1000ms. The position of the images was randomly chosen to be either

left or right to the location of the fixation point. When the two pictures disappeared, a probe stimulus (max. 1000ms) appeared immediately in a location corresponding to the centre of one of the pictures that disappeared. Participants were required to press a button if the probe appeared on the left and another button if the probe appeared on the right hand side. The position of food and non-food pictures was balanced across trials. Twelve practice trials were followed by 40 experimental trials. The order of pair presentation was randomised. In line with previous studies, an attention bias score was calculated by subtracting the mean RT of responding to a probe replacing a food picture from the mean RT of responding to a probe replacing a non-food picture. Thus, positive values indicate faster mean latencies (i.e., an attentional bias) for food than for non-food. Only trials with correct responses were considered for analyses. Response latencies shorter than 100ms were discarded (Miller & Fillmore, 2010).

Interventions

Participants in the real ABM condition were given a treatment version of the AAT (Brockmeyer et al., 2015a). This version adopts an implicit learning paradigm by presenting all food pictures in the "push" (i.e., avoid) format so that participants learn to link avoidance movements to visual cues of high calorie food. Participants received ten 15 minute sessions of training over a 4 week period. Participants in the control condition (sham CBM) received the same dosage of the task but were *not* trained to avoid food cues. Instead, they received 10 additional sessions of the assessment version of the task, which requires an equal number of approach and avoidance movements to both food and non-food pictures. The decision to use 10 treatment sessions was based on two reasons: First, previous research has shown 6 training sessions to be the mean optimal dosage for ABM in alcohol dependence although many patients still improve further with more sessions (Eberl et al., 2014). Second, we used 10 sessions over 4 weeks in our previous proof-of-concept study and found this dosage to be effective and feasible (Brockmeyer et al., 2015a). One training session consisted of 264 trials (i.e., 132 food pictures and 132 non-food pictures – previous ABM studies used similar regimes, e.g. Eberl et al., 2013). All sessions took place in dedicated research facilities. An assessor was present throughout the training and assessment sessions to ensure study adherence.

Data analyses

Data quality and completeness were determined by descriptive statistical analyses and graphical methods. Data points were considered outliers if they were more than 3.29 times the SD above or below the mean. These outliers (n=7) were winsorised (replaced by the group mean +/-3.29 times the standard deviation).

To evaluate treatment-specific changes over time, 2 x 2 repeated measures ANOVAs with group as between-subject factor and time as within-subject factor were applied for each outcome variable. Additionally, multiple regression models were used to examine the relationship between changes in the cognitive measures and symptom improvement. A two-tailed significance level of p<.05 was used throughout. Because of the small number of drop-outs (i.e., 11%, see also Figure 1) and because it was reasonable to assume that data were missing completely at random, as indicated by Little's Missing-Completely-At-Random (MCAR) test, $\chi^2(190)=161.69$, $p=.933$, we followed a complete-case analysis approach, which only includes participants with all data points complete. Additionally, we conducted intent-to-treat (ITT) analyses for the clinical outcomes (OBEs and EDE scores).

Results

Comparison of treatment conditions and treatment sites

Table 1 presents baseline demographic and clinical characteristics of the sample. Participants in the two treatment conditions did not differ in any of the variables at baseline except for the EDE score (sham condition < real condition). However, the following analyses take baseline scores of each respective variable into account.

Study sites did not differ regarding any variable at baseline except for EDE scores, $F(2,44)=5.38$, $p=.008$. Participants from Heidelberg and Düsseldorf tended to have lower EDE scores than those from London ($p=.002$ and .053, respectively). There were no significant effects of study site on treatment outcomes (all p>.05).

Effects of treatment

Objective binge eating episodes

We found no significant main effect of group, $F(1,46)=0.47$, $p=.497$, but a significant main effect of time, $F(1,46)=14.24$, $p<.001$, indicating that both groups had fewer OBEs after the training (see Table 2). However, counter to our hypothesis, there was no significant group x time interaction, $F(1,46)=0.44$, $p=.510$, indicating that groups did not differ in the magnitude of change in OBEs. We found similar results when we assessed the number of binge-days instead of the number of OBEs (group x time interaction, $p=.730$). Number of OBEs and number of binge-days at baseline did not moderate the effects of treatment condition on number of OBEs and binge-days at follow-up, respectively, as indicated by the lack of significant interaction effects between baseline values in these parameters and treatment condition (both $p>.11$).

ED psychopathology

We found no main effect of group, $F(1,45)=0.82$, $p=.369$, but a significant main effect of time, $F(1,45)=6.14$, $p=.017$, again indicating that EDE scores in both groups were lower after the training. Providing tentative support for our hypothesis, we found a trend towards a significant group x time interaction effect, $F(1,45)=3.99$, $p=.052$. We found the same result when we controlled for the effects of study site: group x time interaction, $F(1,44)=3.94$, $p=.054$. We followed up this marginally significant interaction effect with a medium effect size by paired samples *t*-tests for each of the groups. In the real ABM group we found a significant reduction of EDE scores from baseline to follow-up, $t(22)=3.28$, $p=.003$ with a large effect size, $d=0.83$ (95% CI: -0.02; 1.69). In the sham ABM group, there was no significant change in EDE scores, $t(23)=0.33$, $p=.744$, $d=0.07$ (95% CI: -0.75; 0.88). Exploratory analyses revealed that the group*time interaction effect was largely driven by greater reductions in the 'Eating Concern' subscale of the EDE ($\eta_p^2=0.104$, i.e., moderate between-group effect size) in the real ABM group (η_p^2 = .000, .003, and .042 for the 'Weight Concern', 'Shape Concern', and 'Restraint' subscales, respectively).

Trait food craving

We found no significant main effect of group, $F(1,44)=1.09$, $p=.303$, but a significant main effect of time, $F(1,44)=19.59$, $p<.001$, indicating that both groups reported lower trait food craving after the training. However, there was no significant group x time interaction, $F(1,44)=0.13$, $p=.716$, indicating that both groups showed comparable reductions in trait food craving.

Food cue reactivity

Again, there was no significant main effect of group, $F(1,46)=0.98$, $p=.329$, but a significant main effect of time, $F(1,46)=10.75$, $p=.002$, indicating that food cue reactivity was reduced in both groups after the training. However, there was no significant group x time interaction, $F(1,46)=1.50$, $p=.227$, indicating that the magnitude of change was comparable in both groups.

Food intake

There was no significant main effect of group, $F(1,45)=0.05$, $p=.820$, and also no significant main effect of time, $F(1,45)=0.08$, $p=.780$, indicating that food intake did not differ between groups and did not change from baseline to post-treatment. There was also no significant group x time interaction, $F(1,45)=0.60$, $p=.443$.

Approach bias towards food

There was no significant main effect of group, $F(1,45)=1.35$, $p=.251$, and no significant main effect of time, $F(1,45)=0.10$, $p=.751$, indicating that approach bias towards food did not differ between

groups and did not change from baseline to post-treatment. There was also no significant group x time interaction, $F(1,45)=0.02$, $p=.894$. Approach bias towards food and change in approach bias towards food from baseline to post-treatment were not correlated with number of OBEs, number of binge-days, EDE score, trait food craving, food cue reactivity, or food intake at post-treatment/follow-up and also not with the magnitude of change in these variables between baseline and post-treatment/follow-up (all $p>.141$).

Attention bias towards food

There was no significant main effect of group, $F(1,47)=0.01$, $p=.936$, and no significant main effect of time, $F(1,47)=3.84$, $p=.056$, indicating that approach bias towards food did not differ between groups and did not change from baseline to post-treatment. There was also no significant group x time interaction, $F(1,47)=0.53$, $p=.471$. Attention bias towards food and change in attention bias towards food from baseline to post-treatment were not correlated with number of OBEs, number of days with OBEs, EDE score, trait food craving, food cue reactivity, or food intake at post-treatment/follow-up and also not with the magnitude of change in these variables between baseline and post-treatment/follow-up (all $p>.141$).

Moderator analyses

None of the demographic and clinical variables at baseline moderated the effect of treatment condition on any of the outcome variables (all p>.109). Likewise, additional psychotherapeutic treatment did not contribute to the change in EDE scores (p=.877).

Intent-to-treat analysis

All analyses conducted on the sample of treatment completers were repeated in the ITT sample by using linear mixed models for each outcome variable, with fixed factors of time, group, and time*group interaction, and a random intercept for subject. Results were consistent with the completer sample. Of note, in the ITT analysis, the group*time interaction effect for ED symptoms is significant. Here we report the group*time interaction effects: OBE, F(1,48.42)=0.21, p=.651; EDE, F(1,47.36)=4.54, p=.038; trait food craving, F(1,47.25)=0.01, p=.907; food cue reactivity, F(1,49.91)=0.87, p=.356; food intake, F(1,49.89)=0.55, p=.460; approach bias, (F1,51.50)=0.06, p=.811; attention bias, F(1,50.30)=0.44, p=.509.

Condition prediction

Of the participants who provided responses ($n=52$), in the real ABM group ($n=25$), 13 participants thought they received the active treatment. In the sham ABM group ($n=27$), 14 participants believed

they received the active treatment. Groups did not differ in the proportion of participants who thought they received the active treatment, $\chi^2(1)=0.08$, $p=.781$ (certainty of guess on a scale from 1-10: 6.54±8.52 vs. 4.96±2.21; $p=.36$). These results bolster confidence that participants did not systematically predict their respective treatment condition. Participants who correctly identified their treatment condition did not differ from those who did not regarding treatment outcomes (all $p>.223$).

Discussion

This is the first study examining ABM in EDs. Our primary hypothesis was not supported as participants receiving real ABM did not show greater reductions in the number of OBEs than participants receiving sham ABM. However, participants in both groups experienced significant reductions in the number of OBEs. In terms of secondary outcomes, we found larger reductions in ED symptoms (i.e., in the EDE global score) in the real ABM condition as compared to the sham ABM condition. In the real ABM group, there was a large effect for the reduction in ED symptoms. In contrast, this effect was negligible in the sham ABM group. Furthermore, we found significant reductions in trait food craving and food cue reactivity in both treatment groups but no group differences regarding the magnitude of this change. Finally, we did not find any significant change in food intake, approach bias, and attention bias towards food in either of the groups. Taken together, the results provide limited support that ABM can reduce ED psychopathology and pose a number of questions regarding both the efficacy of the intervention in clinical ED populations and its underlying mechanisms of action.

Interestingly, the superiority of real ABM in improving ED symptoms was mainly driven by greater reductions in scores on the EDE subscale 'Eating Concern' in the real ABM group. This subscale includes items that assess preoccupation with food and fear of losing control over eating. It appears conceivable that this finding reflects a treatment-specific improvement in eating-related self-efficacy (i.e., the tendency to feel confident in the ability to control one's eating behaviour under any circumstances, e.g. in a negative mood (Glynn & Ruderman, 1986)). Previous research has demonstrated that low self-efficacy contributes to binge eating (Glasofer et al., 2013) and that it can be improved by psychological treatment (Wolff & Clark, 2001). In participants who received real ABM, the trained avoidance behaviour in response to food cues may have contributed to a greater confidence to resist the urge to binge eat.

The overall findings, however, dovetail with the mixed evidence from recent studies for the efficacy of ABM in reducing unhealthy eating behaviour (Becker, Jostmann, & Holland, 2017; Kakoschke, Kemps, & Tiggemann, 2017a). Some studies found that ABM leads to reduced food intake (Fishbach & Shah, 2006; Kemps et al., 2013; Schumacher et al., 2016). However, all of these studies were conducted in student samples, comprised only a single session of training and assessed food intake only directly

after training, and used a control condition in which participants were required to consistently show approach behaviour towards food. Not only would such a control condition be ethically questionable in clinical ED populations (since it may increase approach behaviour towards unhealthy food (Schonberg et al., 2014)), but it also artificially inflates the difference between conditions by shifting response biases into opposite directions (Becker et al., 2017). Studies using a more balanced control condition did not find any significant effects of training on subsequent food consumption (Becker, Jostmann, Wiers, & Holland, 2015).

In our own proof-of-principle study, ABM was associated with significant reductions in ED symptoms, trait food craving and cue-elicited food craving as well as in (the targeted) approach bias towards food in individuals with high levels of trait food craving and sub-threshold ED symptoms (Brockmeyer et al., 2015a). In the present study, participants in both groups experienced significant and large reductions in binge eating, ED symptoms, trait food craving, and food cue reactivity. It is a common finding in the recent literature on different cognitive bias modification programmes that the active control condition produces changes similar to the real treatment (Voogd, Wiers, & Salemink, 2017). One possible explanation is that these changes simply represent regression to the mean (i.e., similar changes might occur without any intervention). To examine whether this is the case or whether both treatments lead to greater changes than no treatment, future studies may employ an inactive (e.g., wait-list) control group. Another explanation for the observed changes in both groups could be non-specific treatment factors that are shared by the real and the sham ABM training. Participants in both conditions were repeatedly shown images of food without being able to eat this food. Thus, binge eating and food craving may have been reduced as a result of food exposure with response prevention (Schyns, van den Akker, Roefs, Hilberath, & Jansen, 2018). Given that participants in both groups were repeatedly shown food cues while they were instructed to not directly look at the food but at the outline of the picture (round/rectangular), it could also be that participants were trained in directing their attention away from food (Giel et al., 2011). Although in the present study attention bias was not associated with treatment outcome, this should be further examined in future research. The dot probe task does not capture early attentional processes as it only takes a "snapshot" of where the attention is directed at 1000 ms after the stimulus has occurred. Future studies may want to use eye-tracking to more closely examine attentional processes during training (Werthmann, Field, Roefs, Nederkoorn, & Jansen, 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013). Furthermore, it is worth mentioning that approx. 50% of participants in both the active and the sham condition believed they had received the active treatment. Thus, the largely comparable decrease in unhealthy eating behaviour in both groups may also be attributable to general placebo effects, to some degree. Finally, future studies may wish to include measures of general or food-specific impulsivity which could be a moderating factor of food-related ABM (Kakoschke, Kemps, & Tiggemann, 2017b).

Only 20% of the participants in the present study were currently receiving psychotherapy. Thus, for the vast majority of participants, the training was a stand-alone treatment. In previous clinical trials, beneficial effects of ABM were mainly found when ABM was provided as an add-on to multimodal inpatient treatment (Eberl et al., 2013; Wiers et al., 2011). Perhaps ABM imparts its beneficial effects better in a context where patients strongly focus on altering their problematic consumption behaviour. Additionally, it might be more difficult to achieve significant changes in eating behaviour as (in contrast to alcohol dependence) abstinence is not possible.

The lack of change in approach bias towards food in the present study and the avoidance (instead of approach) bias towards food at baseline in the sham condition cast doubt on the validity and reliability of the AAT. While the training version of the task seems to work well as an intervention (at least in the domain of alcohol dependence (Eberl et al., 2013; Wiers et al., 2013)), the assessment version might not be best suited to detect existing approach biases. In line with research on the validity of the AAT in the alcohol domain (Kersbergen, Woud, & Field, 2015), we found in a recent study that the AAT is able to indicate an approach bias towards food only if participants are explicitly instructed to respond to the content of the pictures and not to task-irrelevant features such as the outline of the picture (Lender, Meule, Rinck, Brockmeyer, & Blechert, 2018).

The present study has some limitations. Despite the a priori power analysis, the sample size was rather small and not qualified to detect small effects. Furthermore, mixing BN and BED patients may not be optimal given the differences in the nature and severity of symptoms in these two types of EDs (Fitzgibbon & Blackman, 2000). Having said that, we did not find any differences between BN and BED patients in our sample regarding any clinical variable at baseline (all $p>.290$). Furthermore, the time-period of the follow-up assessment was rather short (2 months) impeding conclusions regarding the intervention's long-term effects. Another limitation is that participants were not asked to refrain from eating prior to all treatment sessions. We did ask participants to not eat or drink anything other than water two hours prior to the assessments (including the first and final treatment session). However, it could be that some participants took part in some training sessions shortly after having eaten something (i.e., were satiated). Theoretically, this could have contributed to the largely null effects of the treatment as training avoidance behaviour in response to food cues may only work if the individual is in a state of craving. Future studies on ABM in the eating domain should therefore ensure that participants conduct training sessions in a low energy load state. A final limitation of the present study refers to ABM as a potential contraindication. One may expect that ABM could be contraindicated for patients who show severe dietary restraint (along with binge eating). However, a case-wise analysis of changes in restrained eating (as assessed by the EDE 'Restraint' subscale) revealed that 9 participants showed (numeric) increases in restrained eating after the training, however, 8 of these 9 patients were in the sham condition. Thus, it does not seem that ABM poses any harm to individuals who feature

restrained eating besides having binge eating episodes. Unfortunately, we did not measure BMI during and after treatment, so we cannot rule out the possibility of weight changes during or after the training. Future studies should include body weight assessments post treatment in order to document any changes.

In conclusion, the present study provides limited support for the clinical efficacy of ABM in treating BN/BED. Future studies should examine in more detail the interactive effects of ABM with other treatments, and the mechanisms of action underlying the intervention to gain further insight into how the intervention works, in which context it works, and for whom it works best.

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Table 1. *Demographic and clinical characteristics of the sample*

	real ABM (*n* = 27)	sham ABM (*n* = 26)	Test statistic	*p*
Mean age in years (*SD*)	29.00 (11.43)	30.93 (11.06)	$t(54)=0.64$	.524
Sex: female (%)	25 (89%)	23 (82%)	$\chi^2(1)=0.58$	.445
A-level (%)	27 (96%)	28 (100%)	$\chi^2(1)=1.02$	.313
Diagnosis: BN (%)	18 (64%)	15 (54%)	$\chi^2(1)=0.66$	.415
Current psychotropic medication (%)	1 (4%)	4 (14%)	$\chi^2(1)=1.98$	.160
Current psychotherapy (%)	5 (18%)	6 (21%)	$\chi^2(1)=0.11$	.737
Mean BMI in kg/m^2 (*SD*)	23.59 (5.15)	25.95 (5.91)	$t(54)=1.59$	.117
OBEs during previous 28 days (*SD*)	23.46 (22.89)	20.89 (17.71)	$t(54)=0.47$	.640
Days with OBEs (*SD*)	14.57 (7.27)	13.25 (7.33)	$t(54)=0.68$	.501
EDE global score (*SD*)	3.58 (0.90)	3.00 (0.97)	$t(54)=2.30$	.025
FCQT-R (*SD*)*	4.40 (0.58)	4.18 (0.67)	$t(53)=1.30$	.198
Food cue reactivity (*SD*)*	5.42 (7.27)	8.04 (6.24)	$t(51)=1.41$	.166
Food intake in kcal (*SD*) #*	214.87 (180.09)	269.14 (223.00)	$t(51)=0.98$	.333
Attention bias towards food (*SD*)*	5.01 (20.56)	2.89 (24.49)	$t(48)=0.33$	.742
Approach bias towards food (*SD*)*	13.22 (110.91)	-2.44 (84.99)	$t(52)=0.58$	.563

BN = Bulimia Nervosa; BMI = Body Mass Index; OBE = number of objective binge eating episodes during the last 28 days; EDE = Eating Disorder Examination; FCQT-R = Food Craving Questionnaire-Trait-Reduced; # Food intake was assessed in a Bogus Taste Test; * FCQT-R data were missing from 1 participant, Food cue reactivity and food intake data were missing for 3 participants, attention bias data were missing from 3 participants, and approach bias data were missing for 2 participants due to technical errors

Figure 1. *Consolidated Standards of Reporting Trials (CONSORT) diagram illustrating flow of participants through the study; ABM = approach bias modification*

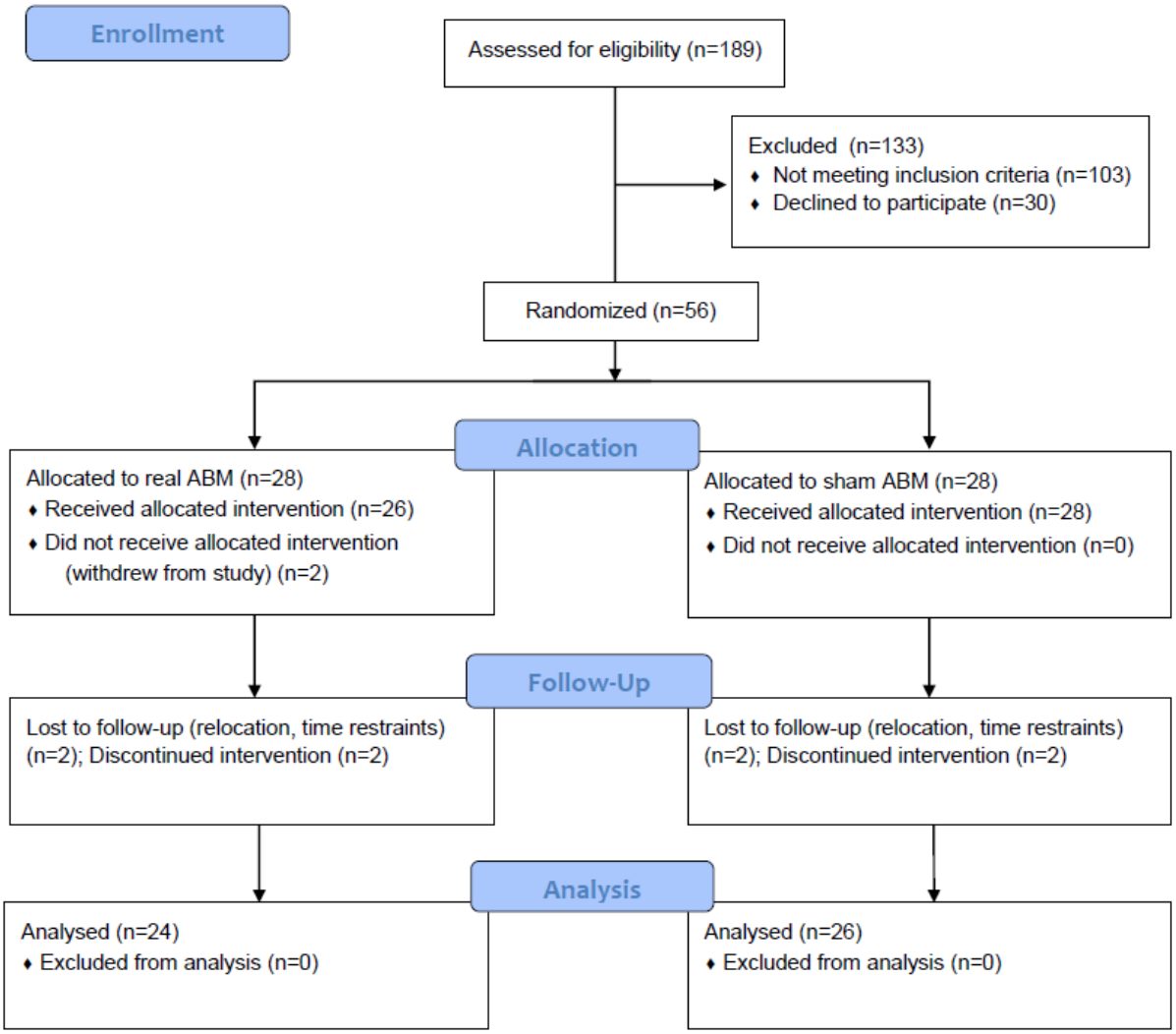

Table 2. *Means and Standard Deviations for Treatment Outcome Measures for Treatment Completers*

	Baseline		Post-treatment/Follow-up	
	real ABM (*n*=24)	sham ABM (*n*=26)	real ABM (*n*=24)	sham ABM (*n*=26)
Variable	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
OBEs	24.38 (24.06)	19.42 (15.01)	14.44 (21.97)[a]	12.36 (17.73)[b]
Eating disorder psychopathology (EDE)	3.40 (0.82)	2.94 (0.93)	2.73 (0.77)[a]	2.82 (1.25)[c]
Trait food craving (FCQT-R)	4.37 (0.57)	4.12 (0.64)	3.91 (0.70)[a]	3.76 (0.84)[d]
Food cue reactivity	5.48 (7.30)[a]	8.56 (6.19)[b]	3.09 (6.40)[a]	3.32 (8.17)[b]
Food intake (kcal)	240.61 (182.33)	274.65 (226.89)[c]	253.78 (259.73)[a]	240.81 (201.67)[b]
Approach bias score	16.52 (116.82)	-11.96 (79.57)[b]	21.09 (131.10)	-7.88 (60.73)[b]
Attention bias score	5.01 (20.56)	2.89 (24.49)	-5.46 (19.38)	-2.22 (23.36)[b]

Note. ABM = approach bias modification; OBEs = number of objective binge eating episodes during the last 28 days; EDE = Eating Disorder Examination; FCQT-R = Food Craving Questionnaire;

[a] Data were missing for one participant, therefore, the sample size for this analysis were real ABM n=23; [b] Data were missing for one participant, therefore, the sample size for this analysis were sham ABM n=25; [c] Data were missing for two participants, therefore, the sample size for this analysis were sham ABM n=24; [d] Data were missing for three participants, therefore, the sample size for this analysis were sham ABM n=23